

**Amendments to the Claims**

This listing of claims will replace all prior versions, and listings, of claims in this application.

1. (Canceled).
2. (Previously presented) A method of effecting an improvement in a standard marker of renal function in a mammal afflicted with acute renal failure, the method comprising administering to said mammal a therapeutically effective amount of an OP/BMP renal therapeutic agent comprising a polypeptide comprising a sequence at least 70% homologous to the C terminal seven-cysteine domain of human OP-1, the sequence of the C terminal seven-cysteine domain of human OP-1 being set forth at residues 330-431 of human OP-1, wherein said renal therapeutic agent:
  - (a) induces chondrogenesis in an ectopic bone assay; or
  - (b) prevents, inhibits, delays or alleviates loss of renal function resulting from acute renal failure in an animal model of acute renal failure;thereby effecting an improvement in a standard marker of renal function in the mammal afflicted with acute renal failure.
- 3-4. (Canceled).
5. (Previously presented) The method of claim 2 or 53, wherein said renal therapeutic agent comprises a polypeptide consisting of at least a C-terminal cysteine domain of a protein selected from the group consisting of a pro form, a mature form, and a soluble form of a polypeptide selected from the group consisting of OP-1, OP-2, OP-3, BMP2, BMP3, BMP4,

BMP5, BMP6, and BMP9.

6. (Previously presented) The method as in claim 5, wherein said renal therapeutic agent comprises a polypeptide consisting of at least a C-terminal cysteine domain of a protein selected from the group consisting of a pro form, a mature form, and a soluble form of human OP-1.
7. (Canceled).
8. (Previously presented) The method of claim 2, wherein said polypeptide has at least 75% homology with an amino acid sequence of a seven-cysteine domain of human OP-1.
9. (Previously presented) The method of claim 2, wherein said polypeptide has at least 80% homology with an amino acid sequence of a seven-cysteine domain of human OP-1.
10. (Canceled).
11. (Previously presented) The method of claim 53, wherein said polypeptide has at least 65% identity with an amino acid sequence of a seven-cysteine domain of human OP-1.
12. (Previously presented) The method of claim 53, wherein said polypeptide has at least 70% identity with an amino acid sequence of a seven-cysteine domain of human OP-1.
13. (Canceled).
14. (Previously presented) The method of claim 2 or 53, wherein said renal therapeutic agent is selected from the group consisting of human osteogenic proteins and human bone morphogenic proteins.
15. (Previously presented) The method of claim 2 or 53, wherein serial determination of BUN in said mammal indicates a rate of increase in BUN of at least 2 to 4 mmol/L/day (5 to 10

mg/dL/day) .

16. (Previously presented) The method of claim 2 or 53, wherein serial determination of BUN in said mammal indicates a rate of increase in BUN of at least 4 to 8 mmol/L/day (10 to 20 mg/dL/day) .
17. (Previously presented) The method of claim 2 or 53, wherein serial determination of serum creatinine in said mammal indicates a rate of increase in serum creatinine of at least 20 to 40  $\mu$ mol/L/day (0.25 to 0.5 mg/dL/day) .
18. (Previously presented) The method of claim 2 or 53, wherein serial determination of serum creatinine in said mammal indicates a rate of increase in serum creatinine of at least 40 to 80  $\mu$ mol/L/day (0.5 to 1.0 mg/dL/day) .
19. (Previously presented) The method of claim 2 or 53, wherein said mammal is afflicted with acute renal failure caused by a pre-renal cause, a post-renal cause, or an intrinsic renal cause.
20. (Previously presented) The method of claim 19, wherein said mammal is afflicted with acute renal failure caused by a pre-renal cause selected from the group consisting of decreased cardiac output, hypovolemia, volume redistribution, and altered vascular resistance.
21. (Withdrawn) The method of claim 19, wherein said mammal is afflicted with acute renal failure caused by a post-renal cause selected from the group consisting of ureteral, pelvic and bladder obstructions.
22. (Withdrawn) The method of claim 19, wherein said mammal is afflicted with acute renal failure caused by an intrinsic

renal cause selected from the group consisting of abnormalities of the vasculature, abnormalities of the glomeruli, acute interstitial nephritis, intratubular obstruction, renal artery occlusion and acute tubular necrosis.

23. (Previously presented) The method of claim 2 or 53, wherein said mammal is a kidney transplant recipient.
24. (Previously presented) The method of claim 2 or 53, wherein said mammal possesses only one kidney.
25. (Withdrawn) The method of claim 2 or 53, wherein said administration is oral.
26. (Previously presented) The method of claim 2 or 53, wherein said administration is parenteral.
27. (Previously presented) The method of claim 2 or 53, wherein said administration is intravenous.
28. (Withdrawn) The method of claim 2 or 53, wherein said administration is intraperitoneal.
29. (Withdrawn) The method of claim 2 or 53, wherein said administration is into the renal capsule.
30. (Withdrawn) The method of claim 26, wherein a stent has been implanted into said mammal for said administration.
31. (Withdrawn) The method of claim 30, wherein said stent is an intravenous stent.
32. (Withdrawn) The method of claim 30, wherein said stent is an intraperitoneal stent.
33. (Withdrawn) The method of claim 30, wherein said stent is a renal intracapsular stent.
34. (Withdrawn) The method of claim 26, wherein said administration

is by an implanted device.

35. (Previously presented) The method of claim 2 or 53, wherein said administration is daily for a period of at least about one week.
36. (Previously presented) The method of claim 2 or 53, wherein said administration is at least once a week for a period of at least about one month.
37. (Previously presented) The method of claim 2 or 53, wherein said renal therapeutic agent is administered at a dosage of about 0.01-1000 µg/kg body weight of said mammal.
38. (Previously presented) The method of claim 37, wherein said renal therapeutic agent is administered at a dosage of about 0.1-100 µg/kg body weight of said mammal.
- 39-52. (Canceled).
53. (Previously presented) A method of effecting an improvement in a standard marker of renal function in a mammal afflicted with acute renal failure, the method comprising administering to said mammal a therapeutically effective amount of an OP/BMP renal therapeutic agent comprising a polypeptide comprising a sequence at least 60% identical to the C terminal seven-cysteine domain of human OP-1, the sequence of the C terminal seven-cysteine domain of human OP-1 being set forth at residues 330-431 of human OP-1, wherein said renal therapeutic agent:
  - (a) induces chondrogenesis in an ectopic bone assay; or
  - (b) prevents, inhibits, delays or alleviates loss of renal function resulting from acute renal failure in an animal model of acute renal failure;

thereby effecting an improvement in a standard marker of renal function in the mammal afflicted with acute renal failure.

54. (Previously presented) The method of claim 53, wherein the standard marker of kidney function is a rate of increase in BUN levels, rate of increase in serum creatinine, static measurement of BUN, static measurement of serum creatinine, glomerular filtration rate (GFR), ratio of BUN/creatinine, serum concentration of sodium ( $\text{Na}^+$ ), urine/plasma ratio for creatinine, urine/plasma ratio for urea, urine osmolarity, or daily urine output.
55. (Previously presented) The method of claim 2, wherein the standard marker of kidney function is a rate of increase in BUN levels, rate of increase in serum creatinine, static measurement of BUN, static measurement of serum creatinine, glomerular filtration rate (GFR), ratio of BUN/creatinine, serum concentration of sodium ( $\text{Na}^+$ ), urine/plasma ratio for creatinine, urine/plasma ratio for urea, urine osmolarity, or daily urine output.
56. (Previously presented) The method of claim 2, wherein administration of the OP/BMP renal therapeutic agent delays the need for, or reduces the frequency of, dialysis treatments of the mammal afflicted with acute renal failure.
57. (Previously presented) The method of claim 53, wherein administration of the OP/BMP renal therapeutic agent delays the need for, or reduces the frequency of, dialysis treatments of the mammal afflicted with acute renal failure.
58. (Previously presented) A method of effecting an improvement in a standard marker of renal function in a mammal afflicted with

acute renal failure, the acute renal failure being one arising from a pre-renal cause of acute renal failure, the method comprising administering to said mammal a therapeutically effective amount of an OP/BMP renal therapeutic agent comprising a polypeptide comprising a sequence at least 60% identical or 70% homologous to the C terminal seven-cysteine domain of human OP-1, the sequence of the C terminal seven-cysteine domain of human OP-1 being set forth at residues 330-431 of human OP-1, wherein said renal therapeutic agent:

- (a) induces chondrogenesis in an ectopic bone assay; or
- (b) prevents, inhibits, delays or alleviates loss of renal function resulting from acute renal failure in an animal model of acute renal failure;

thereby effecting an improvement in a standard marker of renal function in the mammal afflicted with acute renal failure arising from a pre-renal cause of acute renal failure.

59. (Previously presented) The method of claim 58, wherein the pre-renal cause of acute renal failure is selected from decreased cardiac output, hypovolemia, volume redistribution, and altered vascular resistance.
60. (Previously presented) The method of claim 58, wherein the agent is administered—continuously during the period of acute renal failure.
61. (Previously presented) A method of effecting an improvement in a standard marker of renal function in a mammal afflicted with acute renal failure, the acute renal failure being one arising from a pre-renal cause of acute renal failure, the method comprising administering to said mammal a therapeutically

effective amount of an OP/BMP renal therapeutic agent comprising a polypeptide comprising a sequence at least 60% identical or 70% homologous to the C terminal seven-cysteine domain of human OP-1, the sequence of the C terminal seven-cysteine domain of human OP-1 being set forth at residues 330-431 of human OP-1, wherein said renal therapeutic agent:

- (a) induces chondrogenesis in an ectopic bone assay; or
- (b) prevents, inhibits, delays or alleviates loss of renal function resulting from acute renal failure in an animal model of acute renal failure;

wherein the agent is administered continuously during the period of acute renal failure,

wherein the period of acute renal failure lasts from one to three weeks,

thereby effecting an improvement in a standard marker of renal function in the mammal afflicted with acute renal failure arising from a pre-renal cause of acute renal failure.

62. (Previously presented) The method of claim 58, wherein the acute renal failure is characterized by a deterioration of renal function over a period of a few days.
63. (Previously presented) The method of claim 58, wherein serial determination of serum creatinine in said mammal indicates a rate of increase in serum creatinine exceeding 100 mg/dL/day.
64. (Previously presented) A method of effecting an improvement in a standard marker of renal function in a mammal afflicted with acute renal failure, the acute renal failure being one arising from a pre-renal cause of acute renal failure, the method comprising administering to said mammal a therapeutically

effective amount of an OP/BMP renal therapeutic agent comprising a polypeptide comprising a sequence at least 60% identical or 70% homologous to the C terminal seven-cysteine domain of human OP-1, the sequence of the C terminal seven-cysteine domain of human OP-1 being set forth at residues 330-431 of human OP-1, wherein said renal therapeutic agent:

- (a) induces chondrogenesis in an ectopic bone assay; or
- (b) prevents, inhibits, delays or alleviates loss of renal function resulting from acute renal failure in an animal model of acute renal failure;

wherein the mammal is afflicted with osteodystrophy, thereby effecting an improvement in a standard marker of renal function in the mammal afflicted with acute renal failure arising from a pre-renal cause of acute renal failure.

65. (Previously presented) The method of claim 58, wherein the mammal requires continuous hemodialysis sessions.